Case 5: cardiac memory

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A 25-year-old man was seen in the cardiac outpatient clinic with a 10-year history of palpitation. The palpitation, which occurred infrequently and with no obvious precipitant, always started suddenly and was described as fast and regular, associated with a feeling of light-headedness. The attacks usually terminated spontaneously, although on two occasions he had attended casualty following a prolonged episode, where the attacks had been terminated with adenosine. The electrocardiogram (ECG) recorded in the outpatient clinic is shown (*Figure 1*). What is the diagnosis?

DISCUSSION

The ECG shows sinus rhythm with a short PR interval of 80 ms and delta waves that are negative in the inferior leads. The diagnosis is Wolff–Parkinson–White syndrome.

In the normal heart, the only electrical connection between the atria and ventricles is the atrioventricular (AV) node and the His bundle (*Figure 2a*). Slow conduction through the AV node gives rise to the normal PR interval of 120–200 ms. The ventricles are then activated rapidly via the His–Purkinje system giving a narrow QRS complex.

An accessory pathway is a tract capable of electrical conduction across the atrioventricular junction in addition to the AV node or the His bundle. The electrical properties of an accessory pathway usually resemble those of ventricular myocardium rather than those of the AV node, i.e. rapid conduction and a short refractory period.

If an accessory pathway allows antegrade conduction, i.e. from the atria to the ventricles, then the ECG abnormalities of the Wolff–Parkinson–White syndrome are seen. During sinus rhythm, the atrial activation wavefront activates the ventricle through both the AV node or the His bundle and the accessory pathway (*Figure 2b*). Activation through the rapidly conducting accessory pathway results in a short PR interval.

The accessory pathway inserts into ventricular myocardium rather than the His–Purkinje system. Conduction through myocardium occurs more slowly than that through the His–Purkinje system, resulting in slower activation of the ventricles and hence broadening of the QRS complex. When the atrial activation wavefront exits from the AV node, the ventricles are also activated rapidly via the

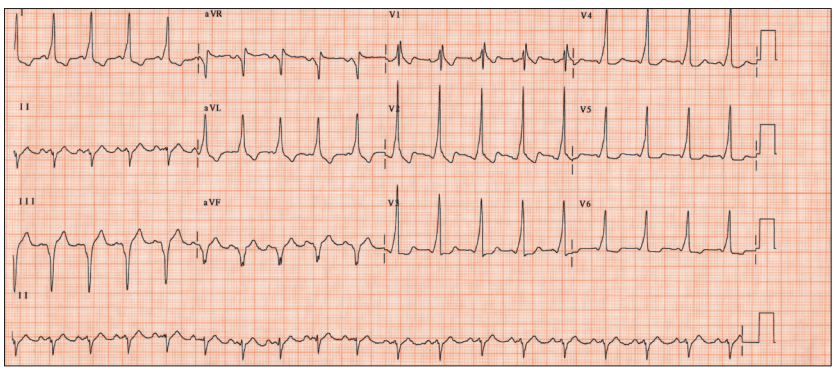

Figure 1. The electrocardiogram during sinus rhythm.

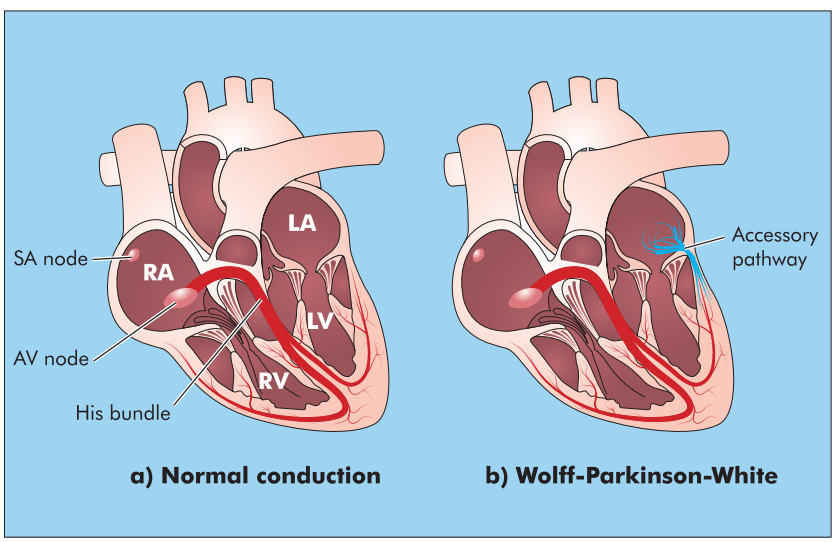

Figure 2. a. Normal conduction. b. Conduction seen in Wolff–Parkinson–White syndrome. AV = atrioventricular; LA - left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle; SA = sinoatrial.

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His–Purkinje system. Thus, during sinus rhythm, the QRS complex is a 'fusion' beat.

In Wolff–Parkinson–White syndrome, the site of earliest ventricular activation is the insertion of the accessory pathway. The activation wavefront proceeds away from this site and therefore gives rise to a negative delta wave in those ECG leads orientated to the accessory pathway. This may be useful in planning a cardiac electrophysiological study with a view to radiofrequency ablation of the accessory pathway. In the example above, an electrophysiological study was performed, and the accessory pathway was localized to the posterior part of the right side of the interatrial septum, activating the inferior surface of the heart first and giving rise to negative delta waves in the inferior ECG leads. Radiofrequency ablation of the accessory pathway was performed.

A routine ECG was performed on the patient's return to the ward (*Figure 3*). The patient was entirely well. In what ways had the ECG changed?

The PR interval had increased to 120 ms, and the delta waves have disappeared. New T wave inversion was present in the inferior leads, while the T waves in the anteroseptal leads were tall and peaked. The ECG demonstrated the phenomenon of cardiac memory (Rosenbaum et al, 1982; Costard-Jäckle et al, 1989). T wave morphology is determined by the vector of ventricular repolarization, which is a function of both the ventricular activation sequence and the cardiac action potential duration.

The relationship between activation sequence and cardiac action potential duration appears to be 'learnt' by the heart during a period with a repetitive activation sequence. When the activation sequence changes suddenly, there is a period lasting from hours to days before the relationship between activation sequence and action potential duration is restored. Although well recognized, the mechanisms responsible for cardiac memory are incompletely understood. Despite this, recognition that ST and T wave abnormalities may occur simply as a result of a change in ventricular activation sequence has important clinical consequences. Examples include:

1. Intermittent ventricular pacing
2. Following ventricular tachycardia
3. Intermittent bundle–branch block
4. Following ablation of an accessory pathway in Wolff–Parkinson–White syndrome.

A further ECG was recorded at the time of outpatient follow-up 2 months later (*Figure 4*). The repolarization abnormalities seen in the immediate post-ablation ECG had resolved, and the ECG was now normal. HM

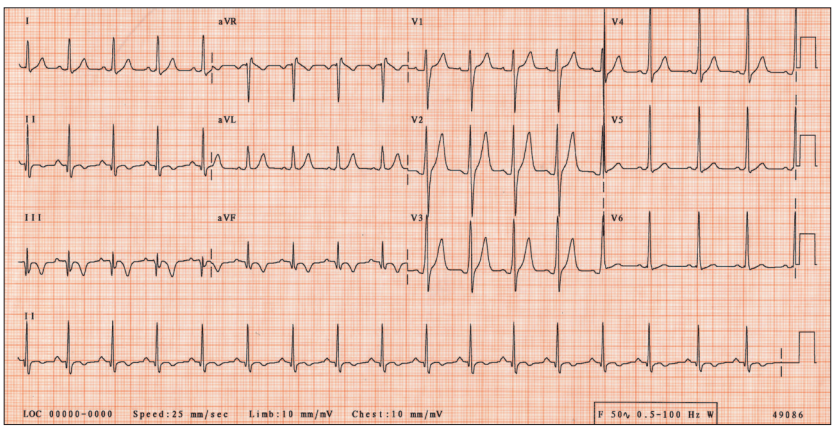

Figure 3. The electrocardiogram immediately after accessory pathway ablation.

Costard-Jäckle A, Goetsch B, Antz M, Franz MR (1989) Slow- and long-lasting modulation of myocardial repolarisation produced by ectopic activation in isolated rabbit hearts: evidence for cardiac 'memory'. *Circulation* **80:** 1412–20

Rosenbaum MB, Blanco HH, Elizari MV, Lázzari JO, Davidenko JM (1982) Electrotonic modulation of the T wave and cardiac memory. *Am J Cardiol* **50:** 213–22

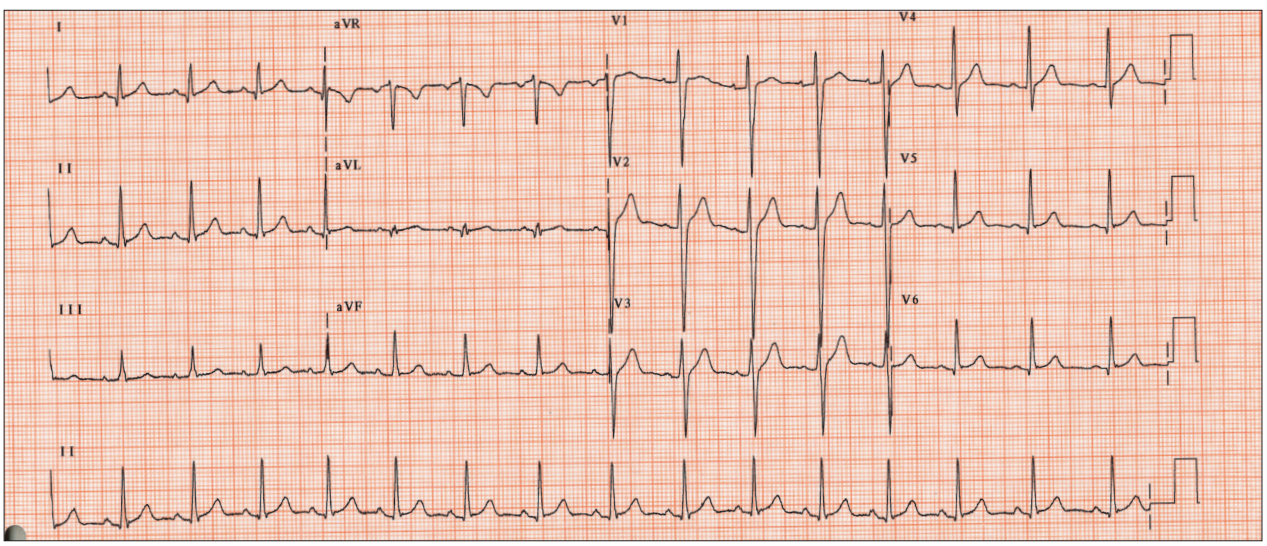

Figure 4. The electrocardiogram 2 months after ablation.